VIRGINIA SURVEY IN CHILDBEARING WOMEN 1989—1995 SUMMARY

Childbearing Women (SCBW) was a cooperative project carried out by three agencies. The HIV Serosurveillance Program of the Division of STD/AIDS, Department of Health, collected the data, entered the data and performed analyses. The Newborn Screening Section of the Division of Consolidated Laboratory Services (DCLS) in the Department of General Services provided dried blood spot samples and tested the samples using EIA and Western Blot technologies. The overall direction, funding, and organization of the program was the responsibility of the Division of HIV/AIDS Prevention of the Centers for Disease Control & Prevention.

Purpose and Process. The public health purpose of the survey was to track HIV infection among childbearing women. Information from the survey was used to help providers, planners and the public understand and combat the HIV epidemic. The survey tested samples of blood drawn from newborns for routine metabolic screening for the presence of maternal antibodies to *human immunodeficiency virus* (HIV). Because a newborn infant's immune system is immature, testing actually detected the mother's HIV status. All samples underwent an initial EIA (enzyme immunoassay) test. Initially positive samples underwent a second EIA test. Repeat positive samples were tested again using Western Blot technology. If an EIA-positive sample was also Western Blot positive, then the sample was considered to be HIV positive. The HIV status of eligible samples was recorded in a confidential database. Although demographic data was also recorded, all personal identifying information was removed from the samples. The program was thus based on a population-based, blinded survey.

Results. This summary outlines survey results between 1989 and 1995; the tables elsewhere in this issue of the *Quarterly Report* provide the frequencies and percentages described. Samples from 1989, 1990, 1991 and 1995 are from a part of the year only. The data for 1992 through 1994 are complete calendar year data.

The results of a clinical trial showing that HIV positive mothers who received zidovudine (AZT) before and during delivery reduced the chance of transmitting the virus to their infants by about 66% caused a re-assessment of public health priorities. The Virginia survey was halted so that scarce resources could be re-directed to enhanced pediatric surveillance. [1, 2, 3, 4]

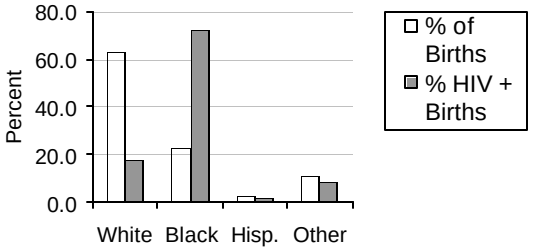

Figure 1: Percent of All Births and Percent of HIV Positive Births, by Race, 1989—1995

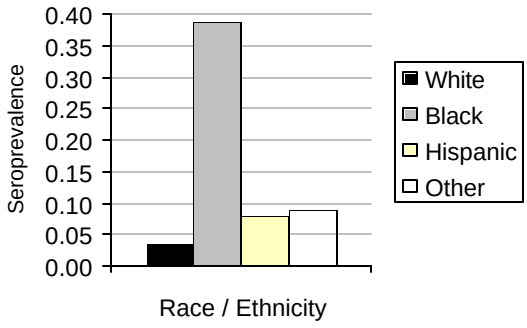

Figure 2: Seroprevalence by Race and Ethnicity, 1989—1995

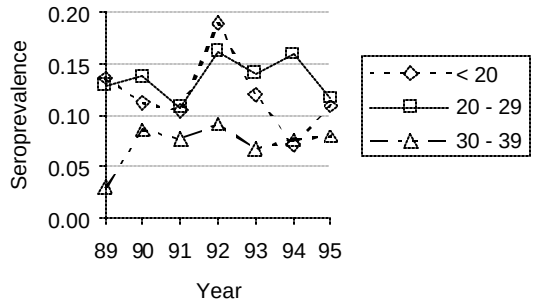

Figure 3: Seroprevalence by Selected Age Group, 1989—1995

Statewide HIV Seroprevalence. HIV seroprevalence for Virginia varied from year to year. In 1989 the rate was 0.10%. In 1990 the rate went up to 0.12%; in 1991 it fell slightly to 0.11%. Seroprevalence reached the highest level in 1992; the rate that year was 0.15%. After 1992, HIV seroprevalence fell each year until it reached 0.11% in 1995. The peak of HIV seroprevalence among all childbearing women occurred in 1992. The cumulative seroprevalence rate for Virginia was 0.12%.

Race and Ethnicity. HIV infections disproportionately affected African American mothers (Figure 1). Although white mothers had 63.3% of all births, they accounted for only 17.6% of all HIV positive tests. African American mothers accounted for 22.6% of all births but had 72.3% of the HIV positive births (Figure 1). This pattern existed in each of the survey years. Hispanic mothers had 2.8% of all births and 1.9% of HIV positive births. Mothers in the 'other' category include Asian and Pacific Islanders, American Indian and Alaskan Natives, others and unknowns.

Differences in seroprevalence rates correspond to the differences in proportions of HIV-positive births (Figure 2). Cumulative seroprevalence among white mothers in the survey was 0.03% while the rate among African American mothers was more than 10 times higher at 0.39%. HIV positivity among Hispanic women was 0.08%. The annual seroprevalence data confirm the overall measures; white mothers had the lowest rates each year, Hispanic women were next lowest and African American women had the highest rates.

Age Group. Childbearing women of all ages in Virginia were infected with HIV. Very young mothers under 15 years old had a cumulative seroprevalence rate of 0.15%. Younger women—those between 15 and 19—tested positive 60 times for an overall seroprevalence rate of 0.12%. Women between 20 and 24 had a higher cumulative seroprevalence rate; the rate in this group was 0.15%. Cumulative seroprevalence among women 25 - 29 was 0.13%, a slight decline from the 20—24 group. HIV infection among those above thirty declined slightly from 0.08% among

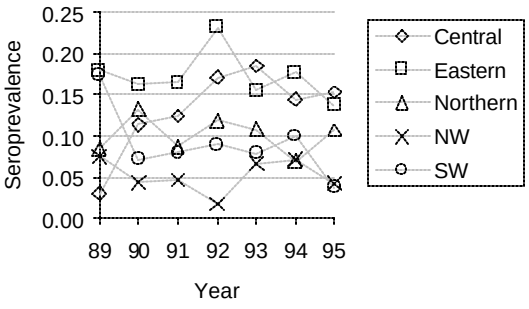

Figure 5: Seroprevalence by Health Region, 1989-1995

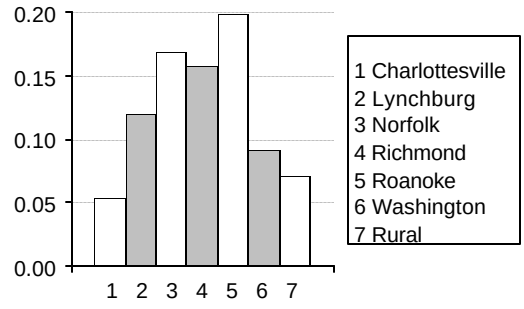

Figure 4: Urban and Rural Seroprevalence (Selected MSAs), 1989—1995

30—34 year-olds to 0.07% among women in the 35-39 group.

Annual seroprevalence figures (Figure 3) show that women in their twenties (20—29) had consistently higher rates than women in their thirties. The peak year in Virginia for the 20—29 year old group was 1992 (0.16%); this was also the case for mothers in the 30—39 group (0.09%). Seroprevalence among women less that 20 years old was more variable from year to year but also followed the pattern of peaking in 1992 (0.19%) followed by a decline in 1993 and 1994.

Regions. Virginia is divided into five health planning regions (Central, Eastern, Northern, Northwest and Southwest). While each region is home to large or major cities, the Northwest and Southwest regions are more rural than the others.

Differences in regional rates have existed since the beginning of the survey. In general, rates in the more urban regions have been higher. During the life of the survey, the Eastern regional rate was highest (0.18%). The Central region was second (0.15%) and the Northern region was third at 0.10%. The Southwest region had a cumulative rate of 0.08% and Northwest was lowest in the state at 0.05%.

Figure 5 shows that the Central and Eastern Regions resemble each other and are distinct from the rest of the state in that seroprevalence in each has exceeded the other regions since 1991 (Central: 0.13%; Eastern 0.17%). The two more rural regions (Northwest and Southwest), generally showed rising seroprevalence between 1990 and 1994. In the Southwest, the rate rose from 0.07% in 1990 to 0.10% in 1994. Northwest was similar; seroprevalence rose from 0.04% in 1990 to 0.07% in 1994. These results suggest that the virus was spreading to residents of the state's more rural areas. The Northern region is an anomaly. Although it is urban like the Central and Eastern regions, seroprevalence rates were lower than Southwest and Northwest between 1991 and 1995.

Metropolitan Statistical Area (MSA). There are eight MSAs either wholly or partially within the state. Norfolk, Richmond and Roanoke had consistently

higher seroprevalence rates than other MSAs in the state. Seroprevalence in Lynchburg (0.26%) was highest in 1993. The very high rate of 0.79% recorded for Roanoke in 1989 was unusual; seroprevalence in Roanoke resembles other high-seroprevalence MSAs in succeeding years. The pattern in Virginia is that urban seroprevalence rates are higher than rural rates. Cumulative seroprevalence for all women living in an MSA was 0.13%. Mothers living in rural locations had a seroprevalence rate of 0.07%. The Virginia portion of the Washington, D. C. MSA is the largest MSA by population in the state (Norfolk is second). Infection among mothers in the Virginia portion the D. C. MSA, however, was more similar to rural rates than it was to other Virginia MSAs. The cumulative rate in Washington MSA was 0.091%.

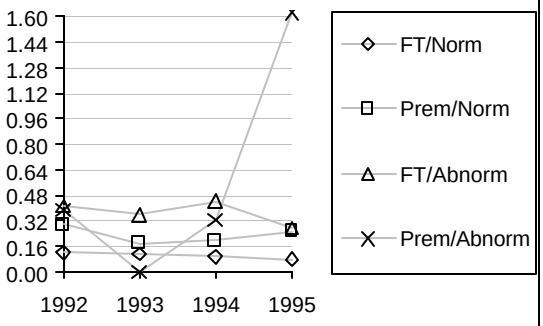

Figure 6: Annual Seroprevalence by Birth Type, 1992-1995

Birth Type. Virginia began collecting type of birth information in 1992. The major categories were full-term normal, premature normal, full-term abnormal and premature abnormal. Abnormal births were considered to be ones that had abnormal results on state-mandated newborn metabolic screening tests (Figure 6).

The majority of births (89.6%) were full-term normal ones and seroprevalence in this category steadily declined from a high of 0.12% in 1992 to 0.08% in 1995. The seroprevalence rate for full-term normal births was generally lower than the rates found in all other groups. Premature normal births had higher seroprevalence rates than full term normal ones; the cumulative rate in this category was 0.23%. African American mothers are highly over-represented in this category with 95% of all positive tests but only 28.6% of all births and a seroprevalence rate of 0.77%. The cumulative rate for white mothers in this group (2.4% of all positive tests and 61.4% of all births) was 0.01%.

The same pattern of African American over-representation occurs among full-term abnormal and premature abnormal births. Of 58 full-term abnormal HIV positive births, 45 (77.6%) were to African American mothers; their cumulative seroprevalence rate was 0.44%. White mothers in this group had seven positive births (12.1%) but accounted for only 21.2% of births in the group (the category includes sickle-cell abnormality, thus naturally increasing the chance that African American mothers will be included). The 0.28% seroprevalence rate among Hispanic mothers in this category is the highest rate in the Hispanic population but the small number of births (355) makes meaningful comparison difficult.

A small number (2,130) of births were both premature and abnormal. Twelve of these

births tested positive for a seroprevalence rate of 0.56%. In this category, African American mothers accounted for the largest number of HIV positive births (eleven, or 91.7%) and the highest percentage of births (32.4%). Seroprevalence among black mothers in this category was 0.89%; this is the highest cumulative rate of all birth types. The overall white rate of 0.15% was also relatively high but the number of births is not large so this result cannot be generalized.

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[1] Conner, EM, Sperling, RS, Gilber, R, et.al. (1994) Reduction of maternal-infant transmission of human immunodeficiency virus type 1 with zidovudine treatment. *New England Journal Of Medicine* 331:1173-80.

[2] Scarlatti, G. (1996) Paediatric HIV infection. *The Lancet* 348:863-868.

[3] Domachowske, J.B. (1996) Pediatric human immunodeficiency virus infection. *Clinical Microbiology Reviews* 9(4):448-468.

[4] Centers for Disease Control and Prevention. (1997) Update: Trends in AIDS incidence, deaths, and prevalence - United States, 1996. *MMWR Morbidity and Mortality Weekly Report* 46(8):165-173.